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INTERNATIONAL PRELIMINARY EXAMINATION REPORT

(PCT Article 36 and Rule 70)

Applicant's or agent's file reference P32800A/CMU/MCM	FOR FURTHER ACTION	See Notification of Transmittal of International Preliminary Examination Report (Form PCT/IPEA/416)
International application No. PCT/GB 03/05163	International filing date *(day/month/year)* 26.11.2003	Priority date *(day/month/year)* 27.11.2002
International Patent Classification (IPC) or both national classification and IPC C07K16/30		
Applicant CANCER RESEARCH TECHNOLOGY LIMITED et al.		

1. This international preliminary examination report has been prepared by this International Preliminary Examining Authority and is transmitted to the applicant according to Article 36.

2. This REPORT consists of a total of ~~15~~ 14 sheets, including this cover sheet.

   ☒ This report is also accompanied by ANNEXES, i.e. sheets of the description, claims and/or drawings which have been amended and are the basis for this report and/or sheets containing rectifications made before this Authority (see Rule 70.16 and Section 607 of the Administrative Instructions under the PCT).

   These annexes consist of a total of 4 sheets.

3. This report contains indications relating to the following items:

   - I ☒ Basis of the opinion
   - II ☐ Priority
   - III ☒ Non-establishment of opinion with regard to novelty, inventive step and industrial applicability
   - IV ☐ Lack of unity of invention
   - V ☒ Reasoned statement under Rule 66.2(a)(ii) with regard to novelty, inventive step or industrial applicability; citations and explanations supporting such statement
   - VI ☐ Certain documents cited
   - VII ☐ Certain defects in the international application
   - VIII ☐ Certain observations on the international application

Date of submission of the demand	Date of completion of this report
13.05.2004	02.03.2005
Name and mailing address of the international preliminary examining authority: European Patent Office D-80298 Munich Tel. +49 89 2399 - 0 Tx: 523656 epmu d Fax: +49 89 2399 - 4465	Authorized Officer Celler, J Telephone No. +49 89 2399-7336

INTERNATIONAL PRELIMINARY EXAMINATION REPORT

International application No. PCT/GB 03/05163

I. Basis of the report

1. With regard to the **elements** of the international application *(Replacement sheets which have been furnished to the receiving Office in response to an invitation under Article 14 are referred to in this report as "originally filed" and are not annexed to this report since they do not contain amendments (Rules 70.16 and 70.17))*:

Description, Pages

1-45 as originally filed

Claims, Numbers

1-20 received on 02.02.2005 with letter of 02.02.2005

Drawings, Sheets

1/2-2/2 as originally filed

2. With regard to the **language**, all the elements marked above were available or furnished to this Authority in the language in which the international application was filed, unless otherwise indicated under this item.

   These elements were available or furnished to this Authority in the following language: , which is:

   ☐ the language of a translation furnished for the purposes of the international search (under Rule 23.1(b)).

   ☐ the language of publication of the international application (under Rule 48.3(b)).

   ☐ the language of a translation furnished for the purposes of international preliminary examination (under Rule 55.2 and/or 55.3).

3. With regard to any **nucleotide and/or amino acid sequence** disclosed in the international application, the international preliminary examination was carried out on the basis of the sequence listing:

   ☐ contained in the international application in written form.

   ☐ filed together with the international application in computer readable form.

   ☐ furnished subsequently to this Authority in written form.

   ☐ furnished subsequently to this Authority in computer readable form.

   ☐ The statement that the subsequently furnished written sequence listing does not go beyond the disclosure in the international application as filed has been furnished.

   ☐ The statement that the information recorded in computer readable form is identical to the written sequence listing has been furnished.

4. The amendments have resulted in the cancellation of:

   ☐ the description, pages:

   ☐ the claims, Nos.:

   ☐ the drawings, sheets:

5. ☐ This report has been established as if (some of) the amendments had not been made, since they have been considered to go beyond the disclosure as filed (Rule 70.2(c)).

   (Any replacement sheet containing such amendments must be referred to under item 1 and annexed to this report.)

6. Additional observations, if necessary:

   see separate sheet

III. Non-establishment of opinion with regard to novelty, inventive step and industrial applicability

1. The questions whether the claimed invention appears to be novel, to involve an inventive step (to be non-obvious), or to be industrially applicable have not been examined in respect of:

   ☐ the entire international application,

   ☒ claims Nos. 17-20(IA)

   because:

   ☒ the said international application, or the said claims Nos. 17-20(IA) relate to the following subject matter which does not require an international preliminary examination (specify):

   see separate sheet

   ☐ the description, claims or drawings *(indicate particular elements below)* or said claims Nos. are so unclear that no meaningful opinion could be formed *(specify)*:

   ☐ the claims, or said claims Nos. are so inadequately supported by the description that no meaningful opinion could be formed.

   ☐ no international search report has been established for the said claims Nos.

2. A meaningful international preliminary examination cannot be carried out due to the failure of the nucleotide and/or amino acid sequence listing to comply with the standard provided for in Annex C of the Administrative Instructions:

   ☐ the written form has not been furnished or does not comply with the Standard.

   ☐ the computer readable form has not been furnished or does not comply with the Standard.

V. Reasoned statement under Article 35(2) with regard to novelty, inventive step or industrial applicability; citations and explanations supporting such statement

1. Statement

Novelty (N)	Yes:	Claims	1,11-13,15-20
	No:	Claims	2-10,14
Inventive step (IS)	Yes:	Claims	11-13
	No:	Claims	1-10,14-20
Industrial applicability (IA)	Yes:	Claims	1-16
	No:	Claims	

2. Citations and explanations

see separate sheet

Re Item I
Basis of the report

The amended set of claims 1-20 submitted with the letter of 02.02.2005 replaces the set of claims 1-27 as originally filed. The support for the amendments, as required by Art. 34.2(b) PCT, in addition to the support indicated in said letter, is found in the application as fallows.

For claim 10, in regard to chemotherapeutic agent, on page 20 of description, line 4. For a pain relief agent, on page 20, line 10. For anti-emetic, on page 20, line 13.

For claim 15, in regard to a pharmaceutically acceptable excipient, diluent or carrier, on page 22, line 24.

For claim 17, in regard to the method of neutralising the complement activation inhibition activity of CD55, on page 9, line 27 to page 10, line 8.

The amended set of claims 1-20 does not comprise any amendments that would introduce any subject matter going beyond that disclosed in the international application as filed and therefore are regarded as allowable under Art. 34.2(b) PCT, and in consequence, form the bases for the present IPER.

Re Item III
Non-establishment of opinion with regard to novelty, inventive step and industrial applicability

Claims 17-20 relate to subject-matter considered by this Authority to be covered by the provisions of Rule 67.1(iv) PCT. Consequently, no opinion will be formulated with respect to the industrial applicability of the subject-matter of these claims (Article 34(4)(a)(I) PCT).
For the assessment of the present claims 17-20 on the question whether they are industrially applicable, no unified criteria exist in the PCT Contracting States. The patentability can also be dependent upon the formulation of the claims. The EPO, for example, does not recognize as industrially applicable the subject-matter of claims to the use of a compound in medical treatment, but may allow, however, claims to a known compound for first use in medical treatment and the use of such

a compound for the manufacture of a medicament for a new medical treatment.

Re Item V

Reasoned statement with regard to novelty, inventive step or industrial applicability; citations and explanations supporting such statement

The gist of the present invention is based on a discovery that the per se known monoclonal antibody 791T/36, which is specific for the cell surface antigen CD55, is able to block one of the functions of said CD55. This function being the ability of CD55 to inhibit the activation of the complement cascade. Moreover, as it was known before the priority date which structural parts of CD55 are involved in the binding to the monoclonal antibody 791T/36 (see below:), the assumption that any factor which binds to the same structural region of CD55 would also block the ability of CD55 to inhibit the activation of the complement cascade underlies the idea of the use of any such agent as an inhibitor of sad function of CD55. In turn, the inhibition of the CD55 ability to inhibit the activation of the complement cascade, should result in an increased activation of the complement cascade on the surface of the CD55 expressing caner cells. Cancer cells are known to overexpress CD55.
These logical conclusions are supported by two instances of experimental evidence disclosed as examples in the present patent application.
The first instance is the binding of "naked" monoclonal antibody 791T/36 to the surface of the CD55 expressing cells which results in an increased deposition of C3b, i.e. activated complement element, on the cell surface.
The second instance is the treatment of the cancer patients with the monoclonal antibody 791T/36, which was radio-labelled. This was conducted for diagnostic tumour imaging, however, the treated patients show increased long term survival.

Reference is made to the following documents:

D1: SPENDLOVE IAN ET AL: "Mapping the binding site of a novel antibody to SCR-1 and 2 of CD55" IMMUNOLOGY, vol. 98, no. suppl. 1, December 1999 (1999-12), page 101, XP002279070 Joint Congress of the British Society for Immunology and the British Society for Allergy and Clinica;Harrogate, England, UK; November 30-December 03, 1999 ISSN: 0019-2805

D2: BRADLEY R G ET AL: "A monoclonal antibody directed against SCR1-2 of complement control protein, CD55 enhances C3 deposition and tumour cell lysis." BRITISH JOURNAL OF CANCER, vol. 88, no. Supplement 1, July 2003 (2003-07), page S39, XP002279071 British Cancer Research Meeting 2003;Bournemouth, UK; July 02-05, 2003 ISSN: 0007-0920 (ISSN print)

D3: WO 98/06838 A (HAMANN JOERG ;LIER RENE ANTONIUS WILHELMUS V (NL); STICHTING CENTR) 19 February 1998 (1998-02-19)

D4: WO 00/37489 A (MUELLER HERMELINK HANS KONRAD ;VOLLMERS HEINZ PETER (DE)) 29 June 2000 (2000-06-29)

D5: PIMM M V ET AL: "A BISPECIFIC MONOCLONAL ANTIBODY AGAINST METHOTREXATE AND A HUMAN TUMOUR ASSOCIATED ANTIGEN AUGMENTS CYTOTOXICITY OF METHOTREXATE-CARRIER CONJUGATE" BRITISH JOURNAL OF CANCER, LONDON, GB, vol. 61, no. 4, 1990, pages 508-513, XP009028842 ISSN: 0007-0920

D6: SPENDLOVE I ET AL: "Decay accelerating factor (CD55): a target for cancer vaccines?" CANCER RESEARCH. UNITED STATES 15 MAY 1999, vol. 59, no. 10, 15 May 1999 (1999-05-15), pages 2282-2286, XP002279072 ISSN: 0008-5472

(D6) Spendlove et al, 1999, 2282-2286, discloses the monoclonal antibody 791T/36 as binding to CD55. The exact region of binding is not defied and the medical use of said antibody in treatment of cancer is not disclosed nor suggested. Therefore said document represents a background knowledge for the subject matter of the application.

(D1) Spendlove and Durrant, 1999, 101, discloses that the antibody 791T/36 binds to the SCR-1 and 2 of CD55.

(D5) Pimm et al, 1990, 508-513, discloses that a bispecific monoclonal antibody against methotrexate and a human tumor associated antigen augments cytotoxicity of methotrexate conjugate. As the bispecific monoclonal antibody is based on the 791T/36 monoclonal antibody and retains the ability to bind the CD55 on the cancer cell, it is obvious in light of the above disclosure (D1) that the binding of CD55 must take place via, both, SCR1 and 2 regions. Moreover, according to the protocol used, the bispecific antibody was supplied to the cancer

cells without methotrexate, That means that it was supplied not being bound to any agent having anti-tumor properties, i.e. as a naked binding member. Methotrexate was supplied sequentially to become localised to the cells via binding to the bispecific antibody present already at the cell surface.

Thus, the bispecifc monoclonal antibody of Pimm et al, has all the structural features of the naked binding member of claims 9.
In other words, the naked binding member which binds to both SCR1 and SCR2 for use in the treatment of cancer is known from Pimm et al, which renders claim 9 not new (Art. 33.2 PCT). Clam 9 does not comprise any technical feature that would distinguish the subject matter for which protection is sought in said claim from the subject matter known from the above disclosure. Therefore, the subject matter known from the prior art is embraced in the scope of said claim which renders it not novel.

The objection to the lack of novelty in view of Pimm et al, also applies to the following claims.
The naked binding member of claim 10 is a part of combined preparation for simultaneous, separate or sequential use in treatment of cancer. As according to the protocol disclosed in Pimm et al, the bispecific monoclonal antibody, which has all the technical features of the naked binding member of claims 9 and 10 was used in combination with sequential administration of methotrexate to treat cancer cells, the combined preparation which is characterised by all the technical features of claim 10 is known from said disclosure, which renders claim 10 also not new (Art. 33.2 PCT).

Moreover, as the monoclonal antibody of Pimm et al is taught to be used in further studies in vivo where the human cancer xenografts would be treated in subjects, a pharmaceutical composition for the treatment of cancer, wherein said composition comprises a naked binding member that binds both SCR1 and SCR2 of CD55 and a pharmaceutically acceptable excipient, diluent or carrier, is taught by said disclosure. Thus, a pharmaceutical composition according to claim 15 is not inventive (Art. 33.3 PCT). The pharmaceutical composition taught by Pimm et all, is characterised by all the technical features of the pharmaceutical composition of claim 15 and would be arrived at by the skilled person without inventive activity.

Moreover, the method of treating cancer, which comprises the administration of a therapeutically effective amount of a naked binding member which specifically

binds to SCR1 and SCR2 of CD55 to a mammal in need thereof is also taught by Pimm et al. Said method has all the technical feature of the method of present claim 19 which renders it not inventive (Art. 33.3 PCT).

Here it should be noted that the above claims 9,10,15 and 19 are not in the format of the second medical use, but rather in the format of the first medical use. For that reason only, the consideration of T290/86, cited in the letter of reply dated 02.02.2005 would not apply to said claims. In addition, it is noted that decision of Technical Board of Appeals considers the question whether the application concerned fulfilled the requirements of EPC and not those of PCT. The current preliminary examination procedure is being conducted under the Articles and Regulations of PCT and not EPC.

Consideration of claims in the second medical use format.

Claim 1 has the format of the second medical use claim, which is for use in the preparation of a medicament for the enhancement of complement deposition on a tissue.
Here it is important to note that the enhancement of complement deposition on a tissue or the lack of enhancement thereof is not a disease per se.
The level of complement activation resulting form the application of an agent is rather to be regarded as a pharmacological effect of that agent.
CD55 is expressed also on tissues of organisms existing in a healthy and not only in a disease state. Therefore, the inhibitory action of CD55, which it exerts on complement activation may be involved in a pathological condition, such as cancer, but it cannot be equated with the pathological state. In consequence, the use of an agent for enhancement of complement deposition on a tissue cannot be equated with a medical use. The same consideration applies to claims 4-8.
The formulation of the claims in the second medical use format: "for use in the preparation of a medicament for" may relate only to a treatment of a pathological condition, a group of patients or manner of administration of the medicament.
Again, the enhancement of complement deposition may be involved in pathological condition, however, cannot be equated with a pathological condition, it does not represent a new group of patient and it does not represent a new manner of administration.
Thus, claims 1,4-8 are not regarded as true second medical use-type claims.

Claim 2, for example, is directed to the use for treating cancer. As cancer is a

pathological condition, claim 2 and claim 3 are regarded as a second medical use-type claims.

Due to the fact that claims 1,4-8 are not true second medical use claims, because, they do not relate to the treatment of any clear pathological condition nor group patients nor manner of administration, the scope of protection sought under said claims is not clearly defined in the claims (Art. 6 PCT).

As discussed above, the molecule that is not bound to any agent having anti-tumor properties, that binds however both SCR1 and SCR2 of CD55 is disclosed in Pimm et al. Moreover, said molecule was disclosed as suitable for use in a preparation of a medicament for treatment of cancer, such as colorectal cancer. Thus, the use of a naked binding member which binds to both SCR1 and SCR2 of CD55 in the preparation of a medicament for treating cancer, such as colorectal caner is known in the prior art. Simultaneously, said use of said naked binding member known from Pimm et al falls entirely under the scope of claims 2 and 3. Said claims do not refer to any technical feature that would distinguish the subject matter of said claims from that known from the prior art. Thus, said claims are not new in the sense of Art. 33.2 PCT. It should be noted that in the sense of Art. 33.2 claims are not new as soon as any embodiment that falls under the scope of claims is not new and even if the scope of said claims also embraces further embodiments which are new per se.
Moreover, as the subject matter of claims 4-8 depends on claims 2 and 3, claims 4-8 also embrace in the scope subject matter known form Pimm et al, which renders them not new (Art. 33.2 PCT).

Moreover, as discussed above, the document of the prior art, Pimm et al, discloses a bivalent monoclonal antibody which binds to SCR1 and SCR2 of CD55. Said bivalent antibody is taught to be used in the treatment of cancer in combination with an active agent, wherein said active agent is a chemotherapeutic agent.
It is acknowledged that said disclosure does not mention the enhancement of complement deposition on a tissue. Furthermore, it is acknowledged that, as indicated in the letter from the applicant, dated 02.02.2005, in the disclosure by Pimm et al, on page 511, column 2, paragraph 2, final sentence, it is stated that the addition of the bispecific antibody alone did not affect target cell survival. Nonetheless, this statement relates to the results of treatment of cancer cells in vitro in the absence of complement constituents.

Thus, the effects associated with the inhibition of complement activation are not detectable in that experimental setup in vitro.
Pimm et al teaches, however, on page 513, column 2, that it is envisaged for use in treating cancer in animal models, in vivo.
Thus, the skilled person is motivated by said disclosure to use the molecule of Pimm et al in the treatment of cancer in combination with chemotherapeutic agent.
The skilled person would therefore arrive by routine means only at the use of the bispecifc antibody of Pimm at al, in combination with chemotherapeutic agent, wherein said bispecific antibody is not bound to any agent having anti-tumor activity. The binding of said chemotherapeutic agent would take place sequentially.
At the same time, it appears very likely that the bispecific molecules of Pimm et al, which has all the structural characteristic of the naked binding member of claim 1, would also be associated with the technical effect of enhancing of complement deposition on cancer tissue in the experimental animal model, because, the components of complement would be present in said animal.
In this manner, the skilled person would arrive at the use of the naked binding member, which binds to both SCR1 and SCR2 of CD55, in the preparation of a medicament for treatment of cancer, wherein the naked binding member is not bound to any agent having anti-tumor properties, and wherein the binding is used sequentially with an agent having anti-tumor properties, and wherein the treatment with said naked binding member would very likely enhance complement deposition on the cancer tissue. The latter would be true, regardless of whether the skilled person would be aware of that mechanism of action of said naked binding member or not.
When claim 1 is read in light of the description, e.g. page 19, line 29, ff. or in view of claim 10, it is apparent that the use of claim 1 may be carried out in combination with other anti-tumor agent or agents.

Thus, it appears that the skilled person would arrive at the use of a naked binding member characterised by all the technical features and associated with all the technical effects of claim 1, regardless, whether the mechanism underlying the effect would be known or not. Therefore, in the absence of any evidence to the contrary, the presence of inventive step for claim 1 cannot be acknowledged (Art. 33.3 PCT), because, the use of the bivalent molecule as taught by Pimm et al, would fall under the scope of said claim.

Moreover, as claims 2-7 do not comprise any technical feature that would

distinguish the subject matter taught by Pimm et al from that taught by the present application, the presence of inventive step cannot be acknowledged also for said claims 2-7.

In the same manner, the skilled person would arrive at a method of neutralising the complement activation inhibition activity of CD55 according to claims 17 and 18, which renders them not inventive (Art. 33.3 PCT), because, the methods of said claims do not refer to any technical feature that would distinguish said methods from those arrivable at based on Pimm at al. In this regard it should be noted that a method according to present claims 17 or 18, which in addition comprises a step of assaying the level of complement deposition on the CD55 expressing tissue, for example, would be regarded as inventive, because, the skilled person would not arrive at such a method based on the disclosure of Pimm et al.

Furthermore, claims 14,16 and 20 refer to the naked binding member as defined in any one of claims 1 to 8. This formulation is not clear (Art. 6 PCT), because, claims 1 to 8 do not define the naked binding member per se, but rather the use thereof. Nonetheless, the content of said claims 1 to 8 refer to a naked binding member having the characterised by the following technical features:

a) binding ability to both SCR1 and SCR2 of CD55 (claims 1-8)
b) not being bound to any agent having anti-tumor properties (claims 1-8)
c) being antibody or fragments thereof (claims 4-8)
d) binding to the specified amino acid residues of CD55 (claims 5-8)
e) comprising on or more of CDRs of the antibody or fragment thereof produced by the specified cell line (claims 6-8)
f) being the antibody 791T/36 (claims 7 and 8)
g) comprising at least one human constant region (claim 8)

The naked binding member known from Pimm et al, i.e. the bispecific antibody has the technical features a)-e). Thus, the naked binding member defined in any of claims 1 to 8 is not new. As indicated above, the naked binding member of claims 9 and 10 are also not new. In consequence the naked binding member of claim 14 is also not new (Art. 33.2 PCT). The pharmaceutical composition of claim 16 is not inventive (Art. 33.3 PCT), the same as the pharmaceutical composition of claim 15, already discussed above. The method of claim 20 is also not inventive in light of the fact that the method of claim 17 is not inventive, as also discussed

above.

(D4) WO00/37489 discloses an antibody SC-1 which binds to CD55 and induces cancer cell apoptosis. Said disclosure does not characterise antibody SC-1 in terms of binding to the SCR-1 and 2 region of CD55, however, it is stated there on page 3, line 15 ff. that the antibody binds tumor cell specific-, and in particular, stomach caner cell-specific isoform of CD55, which is not present on a normal tissue. In consequence, the argument provided with the letter of 02.02.2005, that antibody SC-1 cannot bind the same epitope as that of the present application, because, the antibody of the present application is not tumor tissue specific in binding of CD55 is accepted. Thus, D4 is not considered as relating to the naked binding member according to present claims.

The argument provided in the same letter as above, in regard to the disclosure of (D3) WO98/06838, which relates to the method and means of modifying complement activation, that the agents of WO98/06838 are for inhibiting complement activation resulting from the expression of CD55, rather than for enhancing complement activation at the site of CD55 expression are regarded as convincing. Thus, it is considered unlikely that any of the agent of WO98/06838 would fall under the scope of present claims.

Finally (D2) Bradly et al, July 2003, discloses the invention. The document was published after the priority date of the present application but it states that blocking of CD55 function by SCR1-2 targeting monoclonal antibody generates **potential** therapeutic effects. In light of that it is noted, that the level of enabling support for the technical effect of cancer treatment provided by the present application is comparable with the enabling support present in the disclosure of Pimm et al. Thus, said disclosure of the prior art remains highly relevant, as discussed above.

The subject matter of claims 11-13 is regarded as new and inventive, because, it is neither disclosed nor fairly suggested by any of the cited docuemtns of the prior art.

It should be also noted that the present claims do not fulfil the requirements of clarity (Art. 6 PCT) for the following reasons.

Present claim 13 relates to: "the combined preparation according to claim 13". That means that the claim relates to itself in describing said combined preparation.
This results in a lack of clarity of the claim (Art. 6 PCT).

Moreover, said claim 13 reads: "...or an anti-HER2 antibody e.g. Herceptin, Genentech ...". This formulation is unclear, because, the skilled person would not be certain what other examples are intended to be protected.
An alternative, clear formulation of the claim would be: ...or an anti-HER2 antibody, preferably Herceptin.

Claims 9-13,15,17-19 have as an essential technical feature the naked binding member. However, said naked binding member, is not defined as bing not bound to any agent having anti-tumour properties. The absence of that delimiting features results in that the example of an antibody bound to an radioactive agent, which was used for treating patients, presented in the description, does not fall under the scope of said claims, and as such, represents a comparative example only.

Moreover in regard to the remaining claims which refer to the technical feature of the "naked binding member" wherein the naked binding member is defined as being not bound to any agent having anti-tumor properties, the following is noted. The use of the adjective "naked" appears superfluous. The agent of the claim is fully characterised as an agent or a member, which is not bound to any agent having anti-tumor properties. This characteristic appears to represent a clear technical feature. It remains unclear, however, for what purpose, the adjective "naked" is used in said claims. If, on the one hand, it is to represent the same technical feature as not being bound to any agent having anti-tumor properties, than it is superfluous and the claims so lack conciseness and through that clarity. If, on the other hand, the adjective "naked" is to represent a different technical feature than not being bound to any agent having anti-tumor properties, than it is not clear how is being "naked" differs form not being bound to any agent having anti-tumor properties. In consequence, the claims lack clarity (Art. 6 PCT).

In addition, the expression "binding member" is not commonly used in the art. The commonly used expressions would be binding partner or agent, or interaction partner or agent. To fulfill the requirements of clarity the technical terms should be used in the claims in the same manner as is commonly done in the art.

Claims

1. The use of (i) a naked binding member which binds to both SCR1 and SCR2 of CD55 or (ii) a nucleic acid encoding said binding member in the preparation of a medicament for the enhancement of complement deposition on a tissue, wherein the naked binding member is not bound to any agent having anti-tumour properties.

2. The use of (i) a naked binding member which binds to both SCR1 and SCR2 of CD55 or (ii) a nucleic acid encoding said binding member in the preparation of a medicament for treating cancer, wherein the naked binding member is not bound to any agent having anti-tumour properties.

3. The use according to claim 2 wherein the cancer is one or more of colorectal, breast , ovarian, cervical, gastric, lung, liver, skin and myeloid (e.g. bone marrow) cancer.

4. The use according to any one of the preceding claims wherein the binding member is an antibody or a fragment thereof.

5. The use according to any one of the preceding claims wherein the binding member binds to amino acids 83-93and SCR2 amino acids 101-112 and amino acids 145-157 of the

sequences shown in Figure 1b.

6. The use according to any one of the preceding claims wherein the binding member comprises one or more of the CDRs of the antibody, or a fragment thereof, produced by the cell line deposited at ATCC under accession number HB9173.

7. The use according to any one of the preceding claims wherein the binding member is the antibody 791T/36 produced by the hybridoma cell deposited at ATCC under accession number HB9173.

8. The use according to any one of claims 1 to 7 wherein the binding member comprises at least one human constant region.

9. A naked binding member which binds to both SCR1 and SCR2 for use in the treatment of cancer.

10. A naked binding member, which binds to both SCR1 and SCR2 of CD55, and an active agent as a combined preparation for simultaneous, separate or sequential use in the treatment of cancer, wherein said active agent is a chemotherapeutic agent, a pain relief agent or an anti-emetic.

11. The combined preparation according to claim 10, wherein said active agent is a Doxorubicin, taxol, 5-Fluorouracil, Irinotecan or Cisplatin.

12. The combined preparation according to claim 10 wherein said active agent is an antibody.

13. The combined preparation according to claim 13 wherein said active agent is an anti-CD20 antibody; an anti-VEGF antibody; an anti-CD171A antibody; an anti-CEA anti-idiotypic mAb; an anti-HMFG anti-idiotypic mAb; an anti-EGFR antibody, or an anti-HER2 antibody e.g. Herceptin, Genentech (South San Francisco, CA, USA).

14. The naked binding member according to any one of claims 9 to 10, or the combined preparation according to any one of claims 11 to 13 wherein the naked binding member is as defined in any one of claims 1 to 8.

15. A pharmaceutical composition for the treatment of cancer, wherein the composition comprises a naked binding member that binds to both SCR1 and SCR2 of CD55 and a pharmaceutically acceptable excipient, diluent or carrier.

16. The pharmaceutical composition according to claim 15, wherein the naked binding member

is as defined in any one of claims 1 to 8.

17. A method of neutralising the complement activation inhibition activity of CD55, comprising administration of a naked binding member which specifically binds to SCR1 and SCR2 of CD55.

18. A method of enhancing complement deposition comprising administration of a naked binding member which specifically binds to SCR1 and SCR2 of CD55.

19. A method of treating cancer comprising administration of a therapeutically effective amount of a naked binding member which specifically binds to SCR1 and SCR2 of CD55 to a mammal in need thereof.

20. A method according to any one of claims 17 to 19 wherein the naked binding member is as defined in any one of claims 1 to 8.